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# Foreign Animal Disease Report

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United States  
Department of Agriculture

Animal and Plant  
Health Inspection Service

Veterinary Services

Emergency  
Programs



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New Editor

## Emergency Programs Activities

**Field Investigations.** During fiscal year (FY) 1991 (October 1, 1990, to September 30, 1991), veterinarians from the U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), Veterinary Services (VS), and State departments of agriculture conducted 258 investigations of suspicious foreign animal diseases in the United States and Puerto Rico to eliminate the possibility that an exotic disease may have been introduced. These investigations included 102 for vesicular conditions, 22 for swine septicemic conditions, 7 for mucosal conditions, 54 for exotic Newcastle disease in pet birds and poultry, 4 for avian influenza, 34 for encephalitic conditions, and 35 for undesignated conditions. No foreign animal diseases or pests were found except for exotic Newcastle disease in pet birds, which was expeditiously eliminated.

There has been no exotic Newcastle disease in commercial poultry in the United States since 1974.

**Avian Influenza (AI).** The AI surveillance program in live-bird markets in Florida, New England, New Jersey, New York, and Pennsylvania was continued in FY 1991 (see 18-3: 1-2).

Two live-bird markets in Rhode Island were sampled semiannually; four markets in Massachusetts and five in Connecticut were sampled quarterly. Sampling consisted of collecting and culturing cloacal, tracheal, and environmental swabs.

Thirty-eight live-bird markets, including all of the high-risk markets in New York, were sampled in May 1991 by collecting and culturing tracheal and environmental swabs. AI virus types  $H_2N_2$  and  $H_6N_2$  were isolated from separate environmental samples. Neither proved pathogenic in laboratory chickens.

In New Jersey, 25 live-bird markets, 2 dealer premises, and 2 auction markets were sampled. AI virus type  $H_6N_8$  was isolated from a chicken cloacal swab and an environmental swab, and type  $H_2N_2$  was isolated from two guinea fowl swabs and four environmental swabs.

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In February 1991, the State of Pennsylvania sampled all five live-bird markets in Philadelphia, and randomly sampled live-bird markets at Quakertown and, throughout the State, livestock markets that deal in poultry. Approximately 5,000 eggs from laying flocks and 4,000 blood samples that were collected from broiler chickens at slaughter were also included in the survey. AI virus was not isolated from any of the specimens.

State-employed inspectors placed sentinel birds in live-bird markets in Florida. Blood samples are routinely collected from these sentinels and tested for AI antibodies, and cloacal swabs are collected and cultured for AI virus.

The last H<sub>5</sub>N<sub>2</sub> AI virus isolated from chickens in the United States was from swabs collected from sentinel chickens in a live-poultry market in southern Florida during late September 1989 (see 17-4: 4). The virus was identified at the National Veterinary Services Laboratories (NVSL) in October 1989. The virus was not pathogenic to susceptible chickens and chicken embryos. Poultry at the market were depopulated, and the premises were disinfected.

**Secretary's Advisory Committee on Foreign Animal and Poultry Diseases.** The Secretary of Agriculture's Advisory Committee on Foreign Animal and Poultry Diseases met August 27–29, 1991, in Ames, IA. Seventeen members participated, representing the livestock and poultry industries, the American Veterinary Medical Association, State veterinarians, research and academic communities, and consumers. Eighteen resolutions, comments, or recommendations were made, including recommendations on international trade issues, research and diagnostic laboratory facilities, emergency preparedness, communication between APHIS and American Indian tribal officials, frequency of meetings, disease threats, risk assessment, environmental quality, and other concerns. The committee visited the National Animal Disease Center and the NVSL in Ames during the morning of the first day of the meeting. Current issues involving the prevention, recognition, diagnosis, control, and elimination of foreign animal and poultry diseases were reviewed.

Committee members appointed in 1991 are as follows: Phillip E. Bradshaw, Griggsville, IL; Thomas S. Moorman, San Antonio, TX; Bob Brauer, Oakford, IL; Edward T. Braye, Tuskegee Institute, AL; Bernard C. Easterday, Brooklyn, WI; Dan B. Childs, Lake Placid, FL; Raymond Loretto, San Ysidro, NM; H. Steve Conboy, Lexington, KY; Richard H. McCapes, Davis, CA; Arlene H. Ham, Rapid City, SD; Willie M. Reed, Okemos, MI; Cathy A. Johnson–Delaney, Edmonds, WA; Michele C. Turner, Water Valley, TX; Alfred Keating, Arlington Heights, IL; Taylor H. Woods, Cabot, AR; John H. Lang, Stoughton, WI; Victor F. Nettles, Jr., Watkinsville, GA; Arthur V. Tennyson, Northbrook, IL; and Walter C. Stemler, Waterloo, IL.

(Dr. M. A. Mixson, Emergency Programs, VS, APHIS, USDA, Hyattsville, MD 20782, 301-436-8073)

## Foreign Animal Disease Update

This update is presented in a new format that consolidates information from Office International des Epizooties (OIE) bulletins into tables covering May, June, and July 1991. Countries reporting disease outbreaks are listed below the appropriate disease heading (followed by the month/year of the report and total number of outbreaks reported for that time period). The notation "+" indicates that the presence of disease was reported without information on total number of outbreaks. Outbreak number followed by "+" indicates number of outbreaks as well as disease presence.

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### Foot-and-Mouth Disease\*:

#### *Virus Untyped*

Paraguay (4–7/91) 15  
Niger (1–3/91) 4  
Argentina (3/91) 15  
Myanmar (4–6/91) 8  
Pakistan (5&6/91) 5  
Ghana (7/91) 2  
Chad (3–5/91) +  
Brazil (2&3/91) 46  
Hong Kong (2/91) 1

#### *Virus O*

Egypt (5/91) 1  
Morocco (5–7/91) 10  
Syria (5/91) +  
Colombia (4&5/91) 3  
Ecuador (1&2/91) 5  
Saudi Arabia (1,3,6&7/91) +  
Sri Lanka (12/90–3/91) 26  
Turkey (4–6/91) 267  
Oman (3,4&6/91) 121+  
Algeria (4&5/91) 51  
Pakistan (5&6/91) +  
Paraguay (7/91) 4  
Bulgaria (7/91) 1  
Brazil (2&3/91) 2  
Hong Kong (2&6/91) +  
Thailand (1–4/91) 6

#### *Virus A*

Colombia (4&5/91) 27  
Turkey (4–6/91) 16  
Pakistan (5&6/91) +  
Thailand (3&4/91) 7

#### *Virus C*

Brazil (2&3/91) 5

#### *Virus SAT 1*

Zimbabwe (5/91)

#### *Virus SAT 2*

Cote-d'Ivoire (4–6/91) 11  
Mali (2/91) +  
Ethiopia (5/91) +  
Ghana (1–5/91) 43

#### *Virus SAT 3*

Zimbabwe (7/91) +

#### *Virus Asia 1*

Pakistan (4&6/91) +  
Kampuchea (6/91) +  
Thailand (1–6/91) 72

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### Vesicular Stomatitis

#### *Virus Indiana*

Costa Rica (1/91) 1  
Colombia (4&5/91) 19  
Brazil (2/91) 1

#### *Virus New Jersey*

Costa Rica (1&4/91) 1  
El Salvador (1&4/91) 6  
Guatemala (1/91) 3  
Honduras (1/91) 2  
Mexico (4&5/91) 7  
Nicaragua (1&2/91) 4  
Colombia (4&5/91) 21  
Ecuador (3/91) 1

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### Swine Vesicular Disease

None for this period.

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### Rinderpest

Sudan (5/91) 1  
Sri Lanka (3/91) 1  
Uganda (5/91) 3  
Ethiopia (7/91) 1

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### Peste des Petits Ruminants

Cote-d'Ivoire (4&5/91) 1  
Senegal (3–5/91) 3+  
Oman (3/91) 8  
Ghana (4&5/91) 4

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### Contagious Bovine Pleuropneumonia

Namibia (5/91) 1  
Italy (5–7/91) 10  
Cote-d'Ivoire (4&5/91) 4  
Guinea (4–6/91) +  
Niger (1–3, & 7/91) 2  
Portugal (1–4/91) 233  
Ghana (1&2/91) 2  
Chad (5/91) 1

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**Lumpy Skin Disease**

Mozambique (5-7/91) 5  
Zimbabwe (5&6/91) 44  
Senegal (3-5/91) +  
South Africa (4-7/91) +  
Botswana (4-6/91) 1+  
Swaziland (4/91) 7  
Zambia (4&5/91) 26  
Chad (3&5/91) +

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**Rift Valley Fever**

Mozambique (4-7/91) +  
Namibia (4&5/91) +

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**Bluetongue**

United States (3-7/91) +  
South Africa (4-7/91) +

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**Sheep and Goat Pox**

Israel (5/91) 1  
Israel/Occupied  
Territories (5&6/91) 9  
Senegal (3-5/91) 11  
Pakistan (4/91) +  
Turkey (4-6/91) 117  
Algeria (4&5/91) 9  
Oman (3&4/91) 13

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**African Horse Sickness**

Mozambique (4-7/91) +  
South Africa (4-7/91) +  
Namibia (6/91) 1  
Zimbabwe (6/91) 1

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**African Swine Fever**

Mozambique (4-7/91) 2+  
Spain (4-7/91) 82  
Italy (5-7/91) 33

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**Hog Cholera**

Paraguay (5/91) 2  
Yugoslavia (5/91) 4  
Hong Kong (2&4/91) 2  
Italy (5-7/91) 4  
Mexico (4-6/91) 11  
Argentina (3/91) +  
Colombia (4&5/91) 4+  
South Korea (4-6/91) 12  
Sri Lanka (2/91) 3  
Philippines (3-6/91) 3+  
Taiwan (4-6/91) 18  
Czechoslovakia (4-6/91) 4  
Austria (4/91) 1  
U.S.S.R. (6&7/91) 6  
Germany (5/91) 1  
France (7/91) 1  
Brazil (2&3/91) 7

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**Fowl Plague**

Senegal (3-5/91) 3+1  
Pakistan (4/91) +

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**Newcastle Disease**

Egypt (4-6/91) 15  
Mozambique (5-7/91) +  
Japan (4&5/91) 4  
U.K./Northern Ireland (5/91) 1  
Cote-d'Ivoire (4-6/91) +  
Senegal (3/91) +  
South Africa (4&7/91)  
Zambia (4&5/91) +  
Mexico (4-6/91) 7  
Colombia (4&5/91) +  
Pakistan (4/91) +  
Italy (4/91) 1  
Turkey (4-6/91) 9  
Algeria (4/91) 1  
Yugoslavia (4&5/01) 27  
Ghana (5/91) 53  
Brazil (2/91) 3  
Hong Kong (1-4/91) 13

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**Velogenic Viscerotropic  
Newcastle Disease**

United States (5/91) 4  
(pet birds only) \*\*  
Paraguay (3/91) 1  
South Korea (4-6/91) 13  
Myanmar (4-6/91) 11  
Taiwan (4&5/91) 2  
Indonesia (4-6/91) +

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**Bovine Spongiform  
Encephalopathy**

France (5/91) 1  
Switzerland (5-7/91) 3

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**Porcine Reproductive  
and Respiratory Syndrome**

U.K./Great Britain (5/91) 10  
U.S.S.R. (7/91) 1

\* The untyped foot-and-mouth disease reported in Malaysia for November 1990 (see 19-2: 3) was discovered in a quarantine station in normal, routine testing. The affected animals were destroyed. Malaysia reports that it is free of foot-and-mouth disease.

\*\* Velogenic viscerotropic Newcastle disease has not been seen in commercial poultry in the United States since 1974.

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## 245 Venezuela Equine Encephalomyelitis (VEE) Review

In equines, clinical disease resembling VEE has been recognized since 1925. The etiologic agent was isolated from an equine and characterized in 1938. Human disease definitively shown to be caused by VEE was described in Colombia in 1944. Many large epizootics were reported in Colombia, Venezuela, Trinidad, and Peru from 1939 to 1961. The most notable was Venezuela's 1962–63 epidemic, where there were 23,283 human cases reported with 960 neural complications and 156 deaths. The frequency of epizootics since 1925 has been variable, but VEE epizootics generally occur every 6 to 10 years. A major panepizootic/epidemic of VEE occurred in 1969 through 1971. The epizootic was recognized in Ecuador in January of 1969, where VEE virus was isolated from humans, equines, and mosquitoes. The outbreak then spread to the coastal regions of Peru. The same viral variant was introduced into the coastal area between Guatemala and El Salvador in the early summer months of 1969. Despite aggressive quarantine measures and vaccination programs with an attenuated vaccine (TC-83) supplied by the U.S. Army, the disease quickly spread to Honduras and Nicaragua. By November of 1969, VEE had reached southern Mexico. In 1970, the epidemic continued in western Guatemala, central Honduras, and western Nicaragua and spread to northern Costa Rica. In 1971, the outbreak spread to the central part of Mexico and southern Texas. Further spread into the United States was abated by the massive use of TC-83 vaccine and large-scale, aerial spraying for mosquitoes.

Since 1971, epizootic strains of VEE have not been recognized in Central America or the United States, and there have been no further epizootics or epidemics of VEE reported since 1973. There have been reports of cases resulting from equine vaccination programs using both improperly attenuated and inactivated virus vaccines.

### Etiologic Agent

Members of the Venezuelan encephalitis virus complex are single-stranded, enveloped RNA viruses. The virus particle is composed of three structural proteins: a nucleocapsid protein that is responsible for the serological group reactivity and two surface glycoproteins designated E1 and E2. The E2 glycoprotein is responsible for the hemagglutinating activity of the virus, and specific monoclonal antibodies directed to it can identify specific members within the complex.

### Classification

The VEE virus belongs to the family *Togaviridae* and the genus *Alphavirus*. All members of this genus are arthropod borne and exhibit hemagglutinating activity that is used for serological classification. Members of this genus were previously referred to as Group A arboviruses.

The genus *Alphavirus* contains several serotypes, the most notable being Venezuelan encephalitis (VE), western encephalitis (WE), eastern encephalitis (EE), and Semliki Forest (SF). Three of these, VE, WE, and SF, form complexes of serologically related viruses or subtypes.

The VE virus complex contains six subtypes with several variants. Within subtype I (VEE), only IA, IB, and IC have been associated with epizootics and epidemics and are commonly referred to as epizootic strains.

Epidemiologic classification and geographic origin of VEE virus subtypes and variants are indicated in the following table:

Subtype	Variant	Epidemiologic classification	Geographic origin
I. VEE	A	Epizootic/Epidemic	Trinidad
	B	Epizootic/Epidemic	Honduras
	C	Epizootic/Epidemic	Venezuela
	D	Enzootic	Panama
	E	Enzootic	Panama
II. Everglades	—	Enzootic	Florida
III. Mucambo	A	Enzootic	Brazil
	B	Enzootic	French Guiana
	C	Enzootic	Peru
IV. Pixuna		Enzootic	Brazil
V. Cabassou		Enzootic	French Guiana
VI. —		Enzootic	Argentina

Source of the table: Walton, T. E.; Grayson, M. 1989. The Arboviruses: epidemiology and ecology, vol. 4. Boca Raton, FL: CRC Press: 203–231.

Biochemical analysis has shown that IA and IB are the same, and they are now referred to as I-AB in the literature. The other two variants, ID and IE, and subtypes II, III, and IV are maintained in sylvatic cycles involving rodents and *Culex* mosquitoes and are called enzootic strains. Human disease associated with these viruses is commonly “flulike” and self-limited; however, there have been reports of severe cases with fatalities. Clinical disease in equines has not been reported, but many animals have been found to be serologically positive in serological surveys.

## Geographic Distribution

Epizootic VEE has been reported from ecological settings ranging from sea level to altitudes up to 1,200 m. Generally, the regions have well-developed crop-raising or livestock-raising activities and a well-defined rainy season.

VEE epizootics were reported routinely from Venezuela, Trinidad, Colombia, and Ecuador from 1939 to 1973. During 1969 to 1971, the disease was recognized in Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica, Mexico, and southern Texas. There have been no further reports of the disease in any of these areas since 1973. There is serological evidence that epizootic strains may be circulating within equine populations in central Honduras, Belize, and the Yucatan peninsula of Mexico, but no virus has been isolated.

Enzootic VEE occurs primarily in coastal rain forests or swamps that have moderate to high levels of rainfall year 'round. Variety IE occurs along the Atlantic coastal regions extending from southern Panama to central Mexico. Variety ID occurs along the Atlantic coast of Panama and Colombia. Subtype II occurs in the southern part of Florida. Subtype III occurs primarily along the Atlantic coastal regions of Colombia, Venezuela, Guyana, Surinam, French Guiana, and the extreme northeastern part of Brazil. Subtype IV also occurs in the extreme northeastern part of Brazil, but inland.

## Epidemiology

Epizootics of VEE rely on equines as the amplifying host and mosquitoes as the vector. Equines develop very high viremias for 2 to 4 days. Humans, dogs, swine, lagomorphs, and various sylvatic rodents can develop viremias high enough to infect mosquitoes but probably play only a minor role in epizootics. Sheep, cattle, goats, and bats will develop viremias but probably do not play a role in the spread of the



virus. Birds develop a low viremia that can infect mosquitoes, and birds may be responsible for dissemination of the virus.

Many species of mosquitoes have been associated with epizootics, but the principal genera are *Psorophora*, *Aedes*, and *Mansonia*. After a mosquito feeds on a viremic host, the virus must replicate in the mosquito (intrinsic incubation) before it can be transmitted by the feeding mosquito. In tropical temperatures, this process takes from 8 to 12 days. Since equines develop a high viremia, the possibility of mechanical transmission by *Tabanus*, *Chrysops*, *Simulium*, and *Culicoides* is possible. Recent studies have shown that ticks are capable of transmission and maintenance of the virus.

Enzootic VEE is maintained continuously year 'round in a rodent–mosquito–rodent cycle. Many rodent species are involved, and the main vectors are *Culex (Melanconion)* mosquitoes. People and equines may become infected when venturing into these areas but do not play a role in maintenance of the virus.

## **Clinical Features**

After an incubation period of 1 to 5 days, equines develop a fever greater than 39 °C. Typically, this will be the first sign of disease. Detectable viremia precedes the onset of fever by 12 to 24 hours and persists for 2 to 4 days. Epizootic VEE in the equine can result in three clinical syndromes: inapparent infection with only a mild, febrile response; a more generalized form, characterized by fever, anorexia, depression, and sometimes colic and diarrhea; and an encephalitic form with generalized signs of central nervous system (CNS) involvement, including ataxia, circling, head pressing, and hyperexcitability. The onset of encephalitic symptoms usually occurs when the fever is falling and the circulating virus is disappearing. The virus can cross the placenta, and experiments have shown that it can cause abortion.

## **Pathology**

There are no gross or microscopic pathological changes in equines that are considered diagnostic. Gross pathological signs may vary from no visible lesions to extensive hemorrhage and necrosis in CNS tissue. Histological changes are seen principally in the CNS, hematopoietic, and lymphatic organ systems.

## **Diagnosis**

Clinical diagnosis of VEE is difficult. Encephalitis cases can be confused with western or eastern equine encephalitis or other clinical entities. Only in those situations in which geographic and environmental settings are consistent with those of past outbreaks, and the onset of the disease is explosive, can VEE be reasonably suspected.

Definitive diagnosis must be made by either virus isolation or serology. Virus isolation can be made from the brain, whole blood, or pancreas of dying or newly dead animals. However, even with experimentally infected animals, the isolation rate from these tissues is low. Better success is possible by taking blood samples from herd mates, especially those that are pyrexemic. Since virus is shed in the saliva, it may be helpful to take an oral swab also. Identification of the isolate is a lengthy process because it must be done by virus neutralization or kinetic hemagglutination inhibition (HI) test. Since the development of monoclonal antibody libraries to the E2 glycoprotein, this process should be simplified. In many countries, isolation of the etiologic agent is complicated by the lack of laboratories experienced with VEE virus isolation and identification.

Serologic examination is the most effective way to make a timely diagnosis. One acute serum sample and a second sample, taken 10 to 14 days later, should be

collected to measure a rise in serum antibodies. Current serologic tests used are the serum neutralization, complement fixation, and HI tests; all three require a moderately sophisticated laboratory. An immunoglobulin M (IgM) enzyme-linked immunosorbent assay (ELISA) test for VEE has been developed and should simplify the diagnostic process and greatly decrease the time necessary to make a diagnosis. It is also possible to measure IgM from CNS fluid of dead horses using the IgM ELISA test.

Care must be taken in making a diagnosis of epizootic VEE from serologic testing alone. The complement fixation, HI, and ELISA tests will cross-react with antibodies to enzootic strains. The neutralization test is the most specific test, but even with this test there can be cross-reactions that might lead to wrong conclusions.

Several other diseases present clinical signs similar to those of VEE. These include eastern and western equine encephalitis, plant toxicosis, equine infectious anemia, acute babesiosis, African horse sickness, and hepatoencephalopathy. Starvation, botulism, and extreme internal or external parasitism may also produce signs resembling those of VEE.

## **Prevention and Control**

Experience during the epizootic in southern Texas in 1971 has shown that the combination of mass immunization of equines, large-scale vector control (especially aerial ultra-low-volume insecticide applications), and quarantine can be effective in abating epizootics.

The vaccine used in equines during the epizootic of 1969–71 was developed by the U.S. Army Medical Research and Development Command at Fort Detrick, MD, for use in humans. The vaccine was developed using an attenuated strain, TC-83, and proved to be very efficacious. This vaccine is now commercially available and uses the TC-83 seed stock. The vaccine can produce mild, clinical illness with depression and anorexia. It has been used in pregnant mares with no reported problems. Since it is a modified live vaccine, it requires only one inoculation. That dosage probably provides immunity for life.

A trivalent formalin-inactivated vaccine for western and eastern equine encephalitis and VEE is also commercially available. This vaccine is very effective but requires two doses in the initial vaccination program.

## **Summary**

The VEE complex contains many serologically cross-reactive members. Only two of these, subtypes I-AB and I-C (epizootic strains), produce overt clinical illness in equines and are responsible for epizootics and epidemics. All other varieties are enzootic in nature and do not produce detectable clinical disease in equines and usually produce only mild disease in humans. Enzootic varieties are not responsible for epizootics or epidemics. The enzootic strains are common along the Atlantic coastal regions extending from Brazil to Mexico. Because antibodies to the enzootic strains can cross-react with diagnostic tests for VEE, care must be taken in interpreting serologic results when making a diagnosis of VEE.

(R. Ross Graham, D.V.M., Ph.D., Deputy Commander, U.S. Army Medical Research Unit, Republic of Korea, APO San Francisco, CA 96301-0424)

## 245 Focus on Dourine

Under natural conditions, dourine affects only the horse and the ass. The simple epizootiology of this disease and the development of an efficient diagnostic test allowed the eradication of dourine from most parts of the world. Although dourine is currently limited in its geographic distribution, it is still a serious disease of horses. Therefore, vigilance is needed to prevent reintroduction of the disease into areas of the world where it has been eradicated.

Dourine has been variously called *maladie du coit*, covering disease, equine syphilis, genital glanders, breeding paralysis, chancrous epizootic, epizootic paraplegia, genital paralysis, and venereal disease of the horse.

### Etiology

The etiologic agent of dourine is a large, monomorphic protozoan parasite, *Trypanosoma equiperdum*, of the Salivarian group of trypanosomes of the subgenus *Trypanozoon* in the family Trypanosomatidae (fig. 1).

This trypanosome is morphologically similar to *Trypanosoma evansi*, possibly evolving from *T. brucei* through an intermediate stage as *T. evansi*. *Trypanosoma equiperdum* became monomorphic during repeated direct transmission. This trypanosome divides by longitudinal binary fission in various tissue fluids, particularly in subcutaneous urticarial plaques and in the reproductive system.

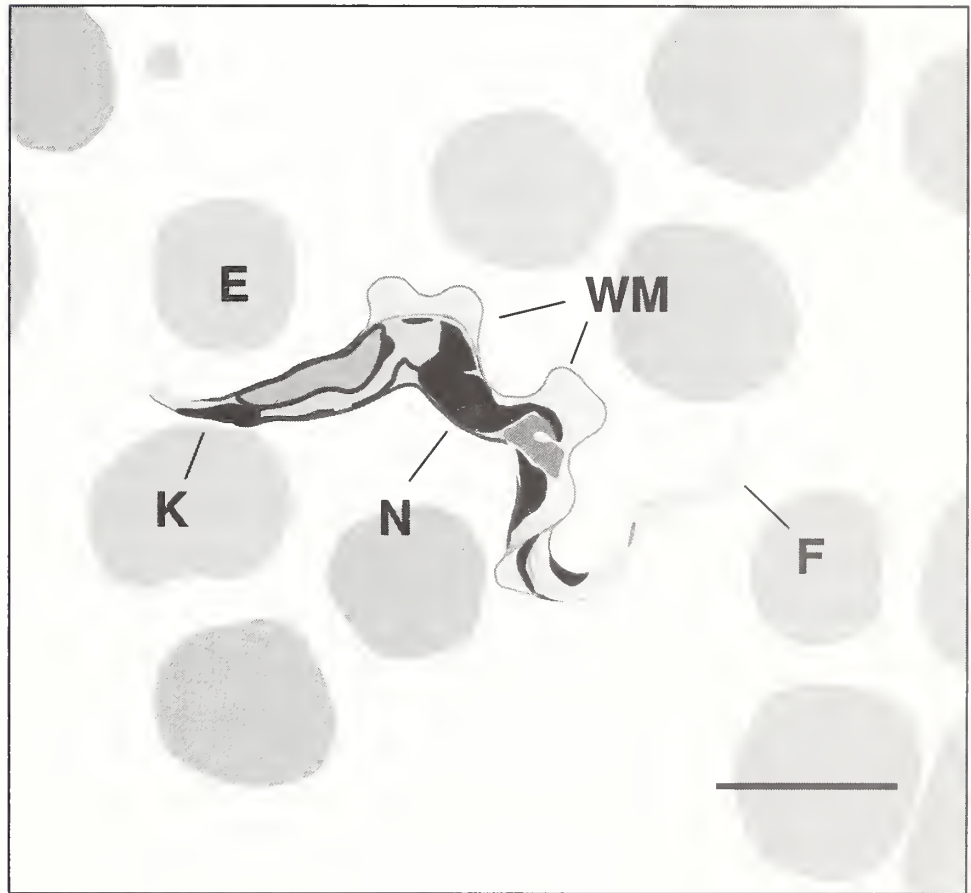


Figure 1—Photomicrograph of *Trypanosoma equiperdum* from a male donkey infected with the American stabilate. E = erythrocytes, F = flagella, K = kinetoplast, N = nucleus, WM = wavy membrane. (Magnification 1000x)

The organism is present in discharges from the genitalia and skin eruptions but does not survive long outside the body.

*Trypanosoma equiperdum* can be microscopically distinguished from other family members by its large size; the presence of a posterior nucleus and subterminal kinetoplast; prominent, undulating membrane with a definite free flagellum; and granular cytoplasm.

## Epidemiology

This trypanosome is chiefly transmitted by coitus, and equids are the natural hosts for this organism. Transmission by biting flies, infective discharges, infected semen, and contaminated artificial insemination equipment is also possible. Strains of *T. equiperdum* with different degrees of virulence exist.

The ass may be an asymptomatic carrier of *T. equiperdum*. Dogs and many laboratory animals can be artificially infected. Guinea pigs exhibit prolonged high parasitemia; rabbits show only chronic disease. The organism remains virulent for the horse in these experimental hosts, which have no role in the epizootiology of dourine.

## History

A disease of antiquity in Asia, dourine was brought to Europe with the importation of Arab breeding stock during the early 1800's. The parasite itself was seen for the first time by Rouget in 1894, but the specific cause of dourine was debated for the next 10 years.

The first cases of dourine described in North America in 1884 were traced to a stallion imported from France in 1882. Although eradicated from the initially affected area of the United States in 1888, the disease had spread with the shipment of several symptomatic, infected animals to other areas. Dourine was most prevalent on the Indian reservations of the Western United States. The disease was eradicated from the United States by 1942.

By the 1900's, the disease had spread to several States and to western Canada. Experimental research concerning the development of a precise method of diagnosis was accomplished at the Quarantine and Research Laboratory, Lethbridge, Canada.

In 1915, E. A. Watson reported (Parasitology 8: 156–183) that the complement-fixation test was a specific, precise, and uniform method of testing for dourine antibody. In 1913, after the utility of complement-fixation had been confirmed in more than 400,000 tests for dourine, the test was accepted for practical use.

## Prevalence

Dourine is prevalent in northern and southern Africa, parts of Asia, and South America. Once widespread in Europe, it is now rarely seen there except in Italy.

In the 1970's, dourine appeared in the Abruzzi region of central Italy. Complement-fixation testing of nearly 5,000 head of horses and donkeys showed that 7.4 percent of these equids were seropositive for *T. equiperdum* (Caporale, V. P.; Battelli, G.; Semproni, G. 1980. Zentralblatt fuer Veterinarmedizin (B) 27(6): 489–498).

Dourine had been reported in southern and southwestern Iran since 1930, but the diagnosis and causative agent had never been confirmed. In 1969, an isolation of *T. equiperdum* from horse blood was confirmed by the Razi Institute of Iran (Khalili, K. 1973. Archives Institut Razi 25: 69–72).



A diagnostic survey in 1985 showed a distinct regional prevalence of dourine in South Africa, despite the low overall incidence of 0.65 percent for animals in the survey (Williamson, C. C.; Herr, S. 1986. *Journal of the South African Veterinary Association* 57(3): 163–165). Because a high proportion of thoroughbreds was included in this study, the relatively high incidence of positive tests in the small sample of nonthoroughbreds led a subsequent investigator to believe that the incidence of dourine in these horses may be considerably higher than the average (Faul, A. 1988. *Journal of the South African Veterinary Association*, 59(1): 1).

## Clinical Signs

The onset of dourine is insidious with an incubation period extending to more than 3 months. Often initial signs are not recognized until the following spring breeding season.

A more severe form of dourine was observed in Asia and Africa. In Europe and the Americas, dourine was more irregular and uncertain in its manifestations.

The disease is marked by stages of exacerbation, tolerance or temporary immunity of uncertain duration, and relapse. These stages may overlap so that disease signs may seem continuous or occur with long intervals in between times when there is no visible sign of disease.

The disease is divided into three well-defined stages: (1) primary—edema, tumefaction, and changes in the genitalia; (2) secondary—plaques and skin eruptions; and (3) tertiary—paralysis, anemia, and cachexia.

A small percentage of infected equids may exhibit ocular signs: photophobia, lacrimation, corneal opacities, keratitis, and changes in the interior of the eye. Stallions may be more prone to these signs. On more than one occasion, corneal opacity was the first sign observed in animals with dourine.

**Primary Stage Signs.** A low, recurrent fever may occur. Initially, there is edema of the genital organs, extending from the scrotum of stallions or udder of mares along the ventral abdominal wall to the chest. Pregnant mares may abort. In males, initial increased libido followed by edema of the prepuce and penis leads to paraphimosis. Genital irritation is evident, leading to frequent attempts at urination. In both sexes, there is hyperemia of the mucous membranes with follicular hyperplasia leading to ulceration and a purulent vaginal or urethral discharge. Regional lymph nodes are inflamed and enlarged.

These swollen, edematous areas pit on pressure but are not hot or painful. The ulcers of the mucous membranes and the colored skin areas of the external genitalia may heal, leaving the characteristic depigmented scars of permanent leukodermic patches.

**Secondary Stage Signs.** The true “plaque” or pathognomonic “silver dollar spot” differs in appearance from the edematous patches of the primary stage. The plaque looks as if a thin metal disc had been slipped under the skin. These plaques of 2–10 cm diameter may appear on any part of the body, particularly over the rib cage and the flanks. The site of a plaque is often conspicuous because of the starring or bristling appearance of the hair coat over the lesion. The average plaque persists for 3 to 5 days. A rapid succession of plaques may appear within a brief period during the disease. Trypanosomes can be found in the serous fluid during this eruptive stage of the swelling.



**Tertiary Stage Signs.** The infected equid shows a rapid, progressive anemia, emaciation, and weakness. The nervous system is severely affected with loss of coordination and paraplegic conditions affecting the hind limbs, lips, nostrils, ears, and throat. The animal walks with a wobbling gait, knuckling over at the fetlock joints of the hind limbs. Muscular atrophy in the gluteal region may be observed. Unilateral facial paralysis of the ear, upper eyelid, and underlip on the same side as an affected limb may be evident. Some animals show generalized or segmental hyperesthesia, which is often followed by decreased cutaneous sensitivity. Proprioceptive deficits may be evident. The affected animal looks dejected. There is gradual progressive paralysis until the animal goes down, unable to rise, and suffers a lingering death unless euthanized.

## **Differential Diagnosis**

Coital exanthema, equine infectious anemia, and purulent endometritis, including contagious equine metritis, should be considered. However, the fully developed clinical picture of dourine is pathognomonic and is not seen in any other disease.

## **Diagnosis**

Provisional diagnosis may be based upon history, lesions, and clinical symptoms. The appearance of plaques or silver dollar spots is pathognomonic.

Detection of trypanosomes by microscopic examination of blood, edematous fluid from the genitalia and urticarial plaques, and vaginal washing is often unsuccessful unless the organisms can be concentrated by centrifugation. Smears of edematous fluid or blood should be Giemsa stained to demonstrate trypanosomes.

Primary isolation of the trypanosome by subinoculation into laboratory rodents is difficult unless specialized routes of inoculation are used or the recipient animals have been splenectomized or immunosuppressed by drug therapy.

The complement-fixation test is a sure and specific method for diagnosing dourine. It is effective for diagnosing asymptomatic carriers, and remains the official certification test for importing equines into the United States. However, there have been problems with anticomplementary results with donkey or mule sera.

The complement-fixation test is still a very efficient diagnostic method except in areas where other trypanosomes of the subgenus *Trypanozoon* occur, because such organisms have antigens in common with *T. equiperdum*. Supplemental tests such as the indirect fluorescent antibody test and the agar gel immunodiffusion test may be used to assess the status of anticomplementary sera.

Additional serologic tests such as the ELISA, radioimmunoassay, and immunoelectrophoresis have been used for diagnosing dourine.

## **Disease Recovery**

Recovery is rare in stallions; in mares, the evolution of the disease is slower and more irregular and uncertain in its result. The recovery rate in mares may be 20 to 30 percent.

## **Therapy**

Treatment is not recommended because eradication of the disease is a more desirable result. The agent is sensitive to drugs, such as quinapyramine sulfate, neosalvarsan, and diminazene aceturate, but results are variable.

Treatment of diseased animals may result in inapparent carriers of the disease.

## **Control**

Treatment is not recommended in a clean geographic area. After official confirmation of dourine in a horse, the breeding of equids in the area should be stopped. All equids in the area should be quarantined. Culling of infected animals is recommended to reduce risk of further disease spread.

No treatment should be attempted in any dourine-free territory (Blaha, T. 1989. Applied veterinary epidemiology. New York: Elsevier: 184–185).

A complete list of references for this article may be obtained by contacting the author.

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## **Subject Index**

A subject index covering articles that appeared in volumes 10 through 19 of the Foreign Animal Disease Report is available upon request.

## **New Editor**

Dr. M. A. Mixson temporarily assumes the editor's role for the Foreign Animal Disease Report with the Spring 1992 issue. Dr. Edwin I. Pilchard, who edited the publication from 1982 to the present issue, retired from Federal service January 3, 1992.

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